

## SKELETAL SYPHILIS IN THE ADULT\*

BY

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Late adult bone syphilis, in which the characteristic lesion is an osteoperiostitis, is now relatively rare in this country. Truog (1943) reported nine cases and said that all acquired syphilitic bone lesions should be referred to as syphilitic osteomyelitis. The end results in the untreated patient may be necrosis, sclerosis, osteoporosis, sequestrum formation, or spontaneous fracture, and any combination of these findings may be present so that a multiplicity of pathological processes may appear in the radiograph.

Gaskell (1951) portrayed radiologically the differential diagnosis of adult bone syphilis from non-syphilitic lesions. He states that unnecessary, sometimes mutilating, operations may be avoided as a result of deliberation upon an x-ray film.

With the increasing use of serological tests in antenatal care, the lesions of congenital bone syphilis, osteochondritis, and periostitis are also comparatively uncommon. However, in a baby with congenital syphilis reported by Marks (1954), the diagnosis was made from positive radiographs.

The following case is reported, because of the extensive skeletal involvement, to stress the importance of radiology in diagnosis.

### Case Report

A married man, aged 47 years, was first seen as an out patient on September 14, 1955, with a complaint of headache for 6 weeks. On further questioning in hospital, he said, "This is not really a headache but a pain in the skull". The pain was mainly over the vertex, constant in nature, and often increasing in intensity during the night. He had obtained some relief from tablets containing aspirin, phenacetin, and codeine, which he had taken in large quantities. In addition, over the past 6 weeks he had noticed a dry cough and he had lost 10 lb. in weight. He gave no history of any previous specific infection.

**Family History.**—Mother and father have no known disease the patient being the only child. Wife is alive and well, with negative Wassermann and Kahn reactions.

Two sons, aged 10 and 8 years, are both in good health with negative Wassermann and Kahn reactions.

### Examination

On admission the patient was a pale, ill-looking man with evidence of loss of weight and cyanosis of the lips, ears, and finger tips; apyrexial and with no obvious dyspnoea.

**Skeletal System.**—No skull or other bony tenderness. There was partial ankylosis of both elbow joints, with limitation of flexion and extension. The right femur and left tibia were grossly thickened, and the right thigh and leg were wasted.

**Cardiovascular System.**—No abnormality. Blood pressure 130/80.

**Respiratory System.**—Signs of fibrosis in the right upper zone.

**Abdomen.**—The spleen was palpable three finger-breadths below the left costal margin and was smooth, firm, and non-tender. The liver was not felt. The testes were noted to be small, but regular in contour with normal sensation.

**Central Nervous System.**—The pupils reacted to light and accommodation. The left knee jerk and both ankle jerks were absent. Plantar responses were flexor. Abdominal reflexes were very brisk and the soles of the feet were extremely sensitive. All modalities of sensation were intact.

### Laboratory Investigations

**Blood.**—Haemoglobin 58 per cent.; Colour index 0.66; White Blood Corpuscles 12,000/c.mm.

Polymorphonuclears	..	79.5 per cent.
Lymphocytes	..	11 per cent.
Monocytes	..	9 per cent.
Eosinophils	..	0.5 per cent.

Sedimentation rate, 45 mm./hr.

Spectroscopy of the blood showed changes, in part at least, due to sulphaemoglobin. This was ascribed to the excessive intake of phenacetin.

Wassermann reaction positive. Kahn test positive.

**Lumbar Puncture.**—Normal dynamics. Clear colourless fluid.

**Cerebrospinal Fluid.**—Wassermann reaction positive. Cells: 2 lymphocytes per c.mm. Protein 70 mg. per cent. Globulin—slight opalescence (Pandy). Lange 000000000.

**Sputum.**—Repeatedly negative for acid-fast bacilli.

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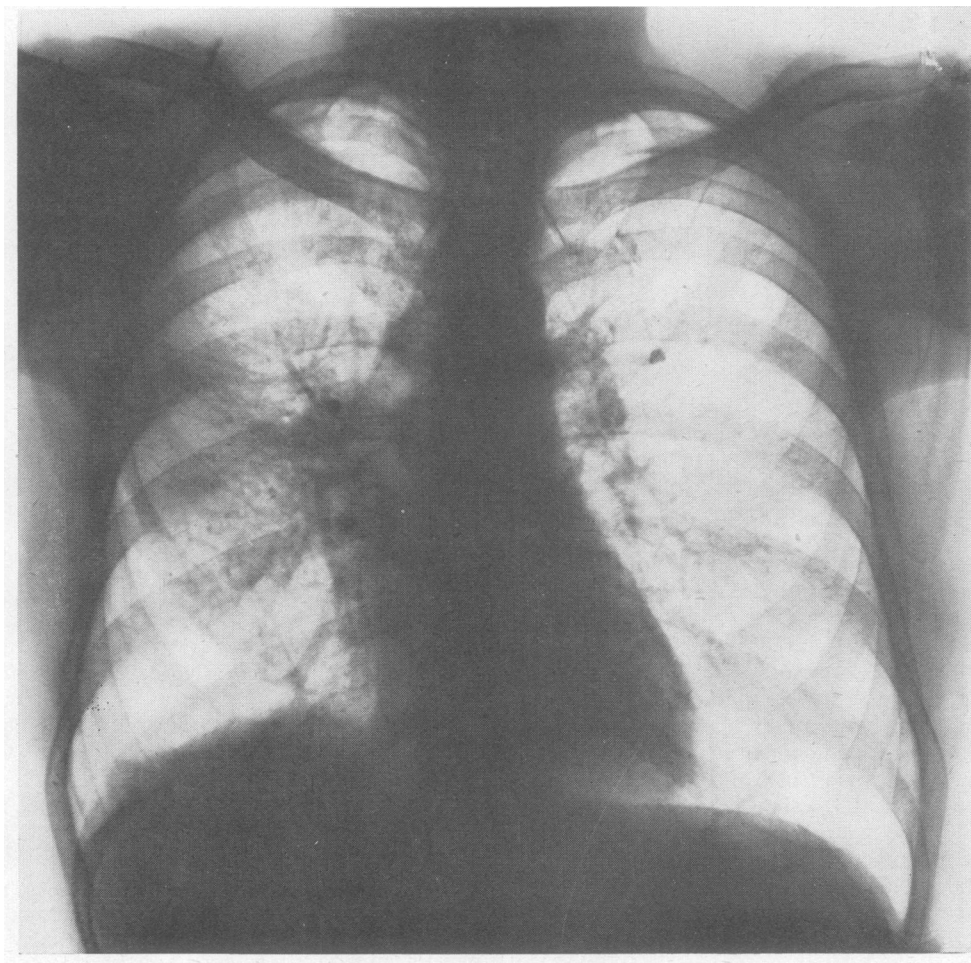


FIG. 1.—X ray of chest.

*Urine.*—No abnormality. Congo Red test within normal limits.

*Liver Function.*—Serum albumin 2.7 g. per cent.  
 Serum globulin 4.7 g. per cent.  
 Thymol turbidity 2.6 units  
 Serum alkaline phosphatase 33 units  
 (King-Armstrong)

Serum bilirubin 0.4 mg. per cent.

Serum acid phosphatase 1.6 units.

Serum calcium 9.5 mg. per cent. Serum phosphorus 3.8 mg. per cent.

*Sternal Marrow.*—Normoblastic with myeloid hyperplasia.

The patient refused to have a bone biopsy.

#### Radiological Examination (See Figs 1–9)

*Chest.*—The mediastinum and right hilum are drawn towards the right and there is fibrosis of the right upper lobe and atelectasis of the right middle lobe (Fig. 1).

Tomography confirmed this and revealed no evidence of bronchial occlusion.

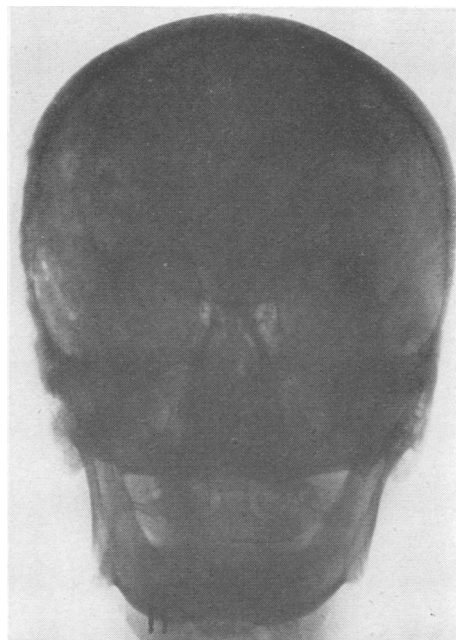
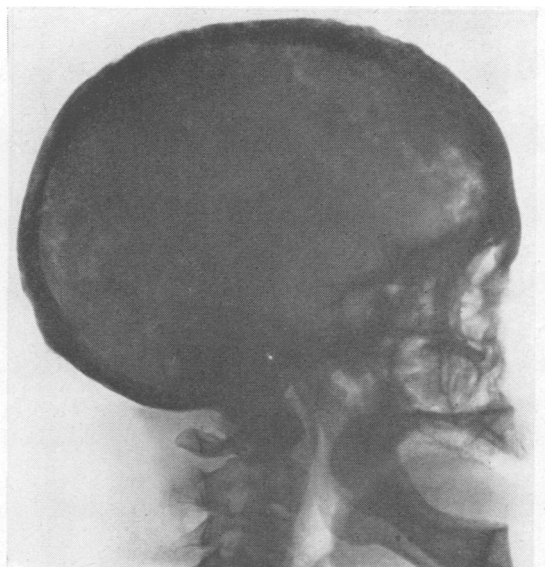
*Skull (a)* Multiple patchy areas of destruction all over the calvarium, involving both tables (Figs 2*a*, *b*, opposite).

(*b*) 3 months after treatment (Figs 3*a*, *b*, opposite).

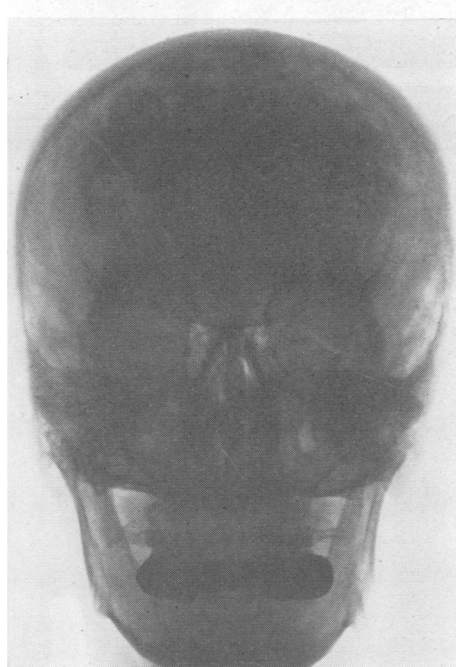
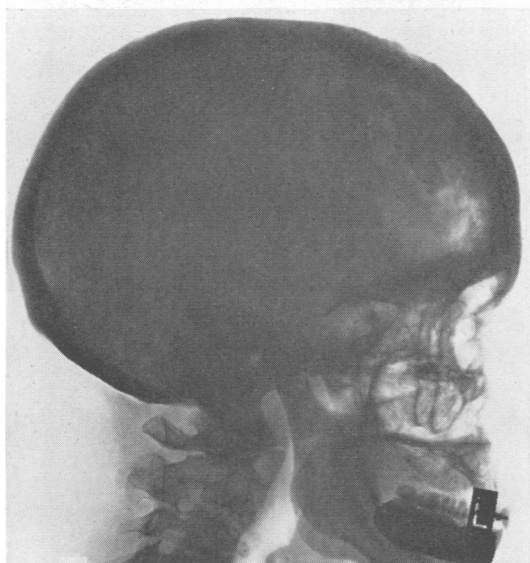
*Femora.*—Numerous areas of cortical destruction and marked widening of the shafts due to severe chronic periostitis (Fig. 4, p. 168).

*Tibiae and Fibulae.*—The left tibia shows marked thickening due to periosteal proliferation, with bone destruction and irregular patchy areas of sclerosis. The right fibula also shows these gross changes, and the right tibia to a lesser extent (Fig. 5, p. 168). Fig. 6 (p. 168) shows the appearances in the right fibula and tibia 5 months after treatment.

*Humeri.*—Both show extensive bone destruction, with periosteal reaction affecting the lower two-thirds of the left humerus; the left acromion process also is involved (Fig. 7, p. 168). The shaft of the right humerus is



FIGS 2a and b.—X ray of skull before treatment.



FIGS 3a and b.—X ray of skull 3 months after treatment.

widened with patchy areas of bone destruction. Both elbow joints are partially ankylosed (Fig. 8, overleaf). The right radius and ulna are involved to a lesser extent.

**Right Ilium.**—This shows alteration in trabeculation and some bone sclerosis close to the anterior superior spine (Fig. 9, p. 169).

**Treatment and Progress.**—All analgesics were discontinued on admission. The patient was given ferrous sulphate and ascorbic acid orally and the anaemia responded well, with a satisfactory reticulocyte rise.



FIG. 4.—X ray of femora.

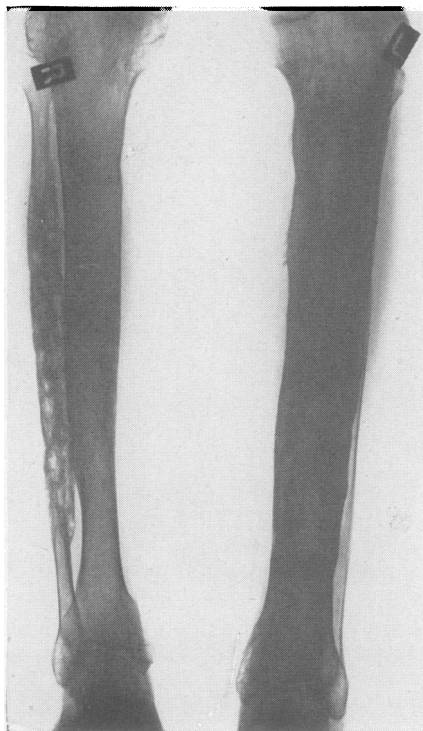


FIG. 5.—X ray of tibiae and fibulae.

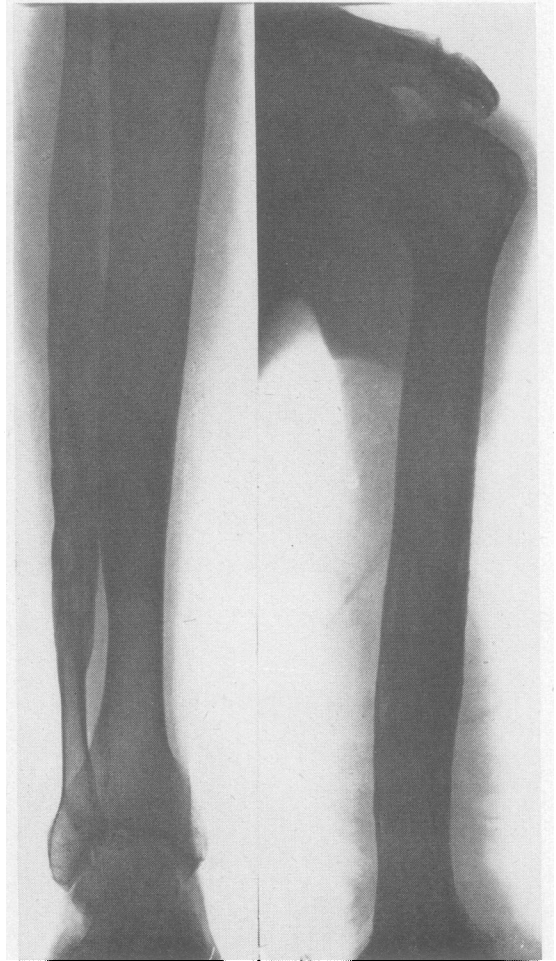


FIG. 6.—X ray of right fibula and tibia 5 months after treatment.

FIG. 7.—X ray of left humerus and acromion process.



FIG. 8.—X ray of both elbow joints.



FIG. 9.—X ray of pelvis (showing right ilium).

Potassium iodide, 20 gr. three times a day, was given for a month with no untoward effects, followed by 12 mega units penicillin intramuscularly over a period of 10 days.

Seven days after commencement of therapy with potassium iodide, the pain in the head disappeared.

Before the patient's discharge from hospital on November 3, 1955, he had gained weight; his haemoglobin had risen to 80 per cent.; the blood sedimentation rate had fallen to 11 mm./hr. and the serum alkaline phosphatase was 24 K.A. units. The spleen was no longer palpable, but the physical signs in the chest and the radiological findings showed no change.

Three months after the beginning of therapy the only significant bony change has been in the skull, where the patchy areas of destruction have become more ill-defined, suggesting that healing is taking place (Figs 3a, b). 5 months after the beginning of treatment there is also improvement in the right fibula (Fig. 6).

#### Comment

Although the aetiology of the pulmonary lesion remains speculative, it is almost certainly not

syphilitic, since there has been no change either in the physical or radiological signs. The nature of the splenomegaly is also not clear, but it is probably related to the initial anaemia, reflecting its extra-haemopoietic function, and its disappearance caused by the iron medication. As there was no clinical enlargement of the liver or gross change in the liver function tests, it seems unlikely that the smooth, non-tender enlargement of the spleen was due to syphilitic cirrhosis or perisplenitis. There was no evidence of amyloid deposition.

At first sight, the presence of head pain, particularly severe at night, with little or no pain elsewhere, in spite of the extensive radiological bone changes, would appear to be an unusual feature. However, it may be that the radiological appearances of the long bones are merely evidence of activity in the past and not of currently active disease. This raises the possibility of congenital infection.

Thomason and Mayoral (1940) treated a patient with syphilitic osteomyelitis with arsphenamine,

bismuth, and potassium iodide followed by prompt relief of symptoms, but state that little or no objective change took place in the osseous lesions since the deformity is usually due to the deposition of new bone, which treatment will not remove.

Keen (1953), in describing his clinical experience with potassium iodide in the treatment of bone syphilis in the Bantu in South Africa, recommends that 90 gr. daily for females and 135 gr. daily for males should be the minimum aimed at, and that the shortest period of treatment should be 6 weeks. He further suggests that a preliminary intensive course of potassium iodide should be administered to all patients showing dense sclerotic bone lesions.

In this case, the diagnosis of syphilis was based not on any one typical finding, but on the widespread distribution of the lesions radiologically, the presence of neurosyphilis, and the confirmatory serological results.

I should like to thank Dr. S. M. Laird for his helpful criticism and advice.

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